Pathology of So-Called Acute Silicosis*

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DURING the past 3 years several reports of so-called "acute silicosis" have appeared in the literature. As far as can be learned Macdonald, Piggot, and Gilder [1] first used the term in a publication describing silicosis in young women employed for 4¼ years or less in packing soap powder. Gerlach and Gander [2] reported a similar condition in a German scouring powder plant and Chapman [3] described its occurrence in an American factory of the same kind. These cases all developed in young women who were said to have been exposed to excessive concentrations of silica dust although no counts of the atmospheric concentrations had been made.

However, the authors were more impressed by the fact that there was free alkali dust in the air of the working places (approximately 25 per cent of such powders are alkalies) than with the high silica concentrations. They concluded that since silica is soluble in an alkaline medium the inhaled material would quickly dissolve to form colloidal silica in the moist lung tissues, and as a result the connective tissues would respond by proliferation much more rapidly than is said to be the case when pure silica is slowly dissolved by the slightly alkaline body fluids.

Kessler [4] reported on a group of sand pulverizers who were alleged to have developed silicosis after exposures as short as 4 months to 1½ years. These were young male negroes. It was stated that the dust concentrations were excessive although the actual figures are not available. The particles were very fine (1 to 5 microns in diameter) and over 99 per cent of them were free crystalline silica. In this group it was assumed that the rapid development of the silicotic process was due solely to the high concentration of dust in the atmosphere. Since the men had not been examined before employment in this plant, they might have had previous exposures to siliceous dust. If so, the silicosis from which they apparently suffered could not be considered acute.

The lack of complete data in these reports raises the question as to whether a chronic disease like silicosis, which in the majority of industries today requires at least 10 or 12 years for its development, and which under the very worst conditions in the past only became recognizable after 3 or 4 years, can under any conditions actually develop to a degree where it can be recognized in a roentgenogram in shorter periods of time. If it does occur, what is its character and what are the conditions responsible for the accelerated reaction? Two factors, the presence of free alkali in association with silica dust, and an excessive concentration of the silica itself, have already been suggested. A

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third factor might be particle size, and a fourth, concomitant infection.

The author has had opportunity to study the lung tissues and in most instances ante-mortem roentgenograms from 15 persons who were said to have developed silicosis after dust exposures of from 8 to 17 months. Of these 9 were employed as laborers and drillers in a tunnel through practically pure quartz; 4 were sand blasters, and 3 were sand pulverizers from the same group reported by Kessler.[4] For the last group the occupational histories are most incomplete and probably inaccurate.

No atmospheric dust counts were available, but from all reports excessive amounts of extremely fine dust were generated in each case. In the tunnel, which was bored without effective ventilation, the men returned to the face immediately after blasting and were required to work with dry drills in an atmosphere so exhausted by gasoline engines that they must have breathed abnormally fast. Such conditions would obviously favor the inhalation of unusually large quantities of very fine dust. The sand blasters also were without effective protection; one man dated the onset of his symptoms from the bursting of a sand hose while cleaning the inside of a large tank. The sand was used over and over until it became so fine as to be no longer effective as an abrasive. In the pulverizing plant, where the sand used by the sand blasters was ground and bagged, there were protective devices but these were apparently not altogether effective. While this information implies excessive concentrations of extremely fine silica particles, the dust exposures cannot be measured quantitatively.

In none of the groups had there been any attempt to examine the men when they were employed. Therefore one is compelled to rely upon the occupational history obtained in all cases after the development of alleged symptoms. Such information must be regarded with suspicion when claims for compensation are pending, as was the case with these individuals. Some of the information was obviously valueless, as in the case of a sand pulverizer with a roentgenogram showing widespread pulmonary disease, who had worked in the plant only 35 days. On the other hand, the age of many in these groups and the conditions under which they were hired precluded any prolonged exposure to silica in previous occupations. Five of the tunnel workmen were under 29; the others, with 1 exception, were under 40. Seven were negroes who claimed to have had no previous experience in mines. Two were white men who had been coal miners for 4 and 7 years respectively. The negroes were reported to be common laborers brought from another state under contract. Most of them were employed at times as drillers, an unusual occupation for unskilled laborers, but it was contended that professional miners would not work under the conditions existing in this undertaking. The sand pulverizers were also young negroes whose previous employment was unknown. The sand blasters were all white, aged 26, 27, and 45 respectively. This evidence for the shortness of the exposure periods is also obviously open to criticism, but even if the exposure was not so brief as claimed in some individual cases, nevertheless it could not have been as long as that in industries usually associated with a silicosis hazard.

Since information as to the exact character and duration of the dust exposure in these cases is so limited, the justification for this presentation might be questioned. Several reasons exist. In the litigation attendant upon the presentation of compensation claims many of these persons were alleged to be suffering from an "acute silicosis." With 3 exceptions these men died from

9 to 18 months after completing very short exposures to pure silica. Few autopsies have been reported upon persons dying during the course of such short, severe exposures and therefore these cases may shed some light upon the nature and extent of the silicotic reaction. While it is known that silicosis is a progressive disease, the amount of progression to be expected within so short a time after the exposures must be limited.

in many of the sections they are extremely numerous. The relative size of the nodules which had developed after various exposures could be more accurately measured. In a group of sand blasters working from 1 to 5 years the outline of the central hyaline portions of various nodules in micro-sections were drawn with a camera lucida. Figure I illustrates representative nodules from 4 different cases. After a 12 months' exposure the nodules measured from 0.3

TABLE I

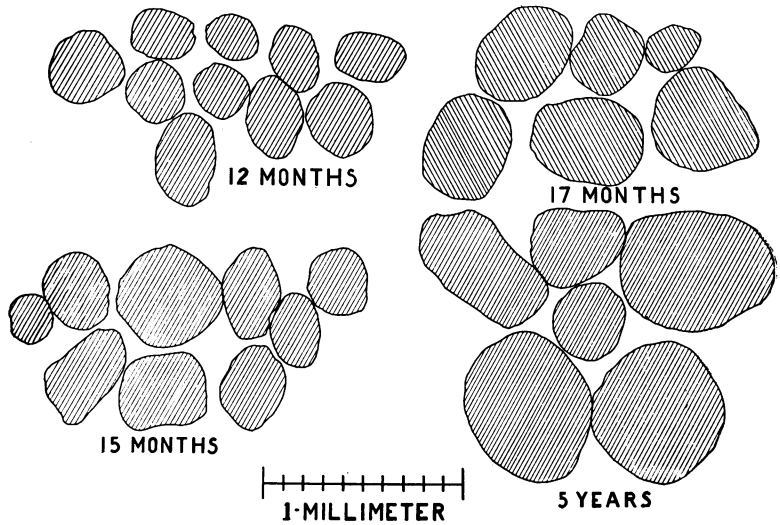
Case No.	Age Color	Occupation	Exposure	Interval to Death	Silicosis	%SiO_2 Dry Lung	Infection	Organisms
63	W 23	Driller	Coal 7 yr. Tunnel 13 mo.	9 mo.	4+ (Anthracosis)	1.98	? TB	O
64	W 46	Laborer	Coal 4 yr. Tunnel 8 mo.	18 "	4+ (Anthracosis)	3.10	Acute and Chr. Non-TB. Pn.	Diplococci
65	C 29	Driller	Tunnel 9 "	15 "	4+	2.78	Caseous Pn.	TBC
66	C 24	Laborer	" 9–10 "	12 "	±	0.875	Caseous Pn.	TBC
67	C 21	"	" 9 "	14 "	+	1.975	TB. Br-Pn.	TBC
68	C 34	"	" 13 "	?	4+	2.77	TB. Br-Pn.	TBC
69	C 29	"	" 13 "	11 "	0	0.84	TB. with Cavity	O
70	C 38	"	" 13 "	13 "	2+	2.58	Cas. Pn. Cavity	O
71	C 37	"	" 13 "	13 "	4+	1.24	? TB	O
34	W 45	Sand blaster	Shop 17 "	21 "	5+	–	Unresolved Pn.	O
36	W 27	"	" 12 "	20 "	4+	–	TB. Pn. Cavity	TBC
47	W 26	"	" 15 "	15 "	3+	–	TB. Pn.	TBC
44	C 28	Sand Pulverizer	35 days?	?	3+	1.81	Caseous Pn.	–
45	?	"	?	?	3+	2.1	Aspiration TB	–
46	C 29	"	3 yrs?	?	3+	2.45	TB. Pn.	–

In all cases but 1 (No. 69) there is definite microscopic evidence of silicosis consisting of characteristic hyaline fibrous nodules. These lesions are definitely smaller but appreciably more abundant than those encountered in the usual run of silicosis cases known to have had more prolonged exposures to dust. It has not been possible to count the actual number of nodules in a given volume of lung tissue, as suitable quantities from corresponding parts of the different lungs were not available; but

to 0.4 mm. in diameter; by 5 years they had attained a diameter of 0.7 to 0.8 mm. In the intervening periods of 15 and 18 months there had been an appreciable increase in size. The nodules in the tunnel workers were approximately 0.4 mm. in diameter. For comparison a few nodules from the lung of a hard rock lead miner were projected in a similar manner. These nodules, too large for illustration, measured from 3 to 4 mm. in diameter. The tracings also demonstrated that all

FIGURE I

Camera Lucida tracings of silicotic nodules at various intervals of exposure. The nodules from the ordinary case of silicosis are larger than the entire area of this figure.



the nodules encountered in any lung are quite uniform in size and that small, apparently fresh nodules are relatively rare after prolonged exposures.

In the cases under discussion the nodules are massed in a compact zone, a centimeter or more in width, beneath the pleura. Along many of the interlobular septa there are wide bands of closely packed nodules which encroach upon the parenchyma on either side. In such formations the lesions are surrounded and embedded in sheets of loose cellular or hyaline connective tissue containing appreciable quantities of fine black dust particles. A few nodules also occur singly or in small clusters throughout the alveolar tissue. When stained by Foot's method for reticulum, the impression is gained that the reaction is relatively young, for black staining reticulum predominates over red stained collagenous elements. In many air spaces are masses of necrotic exudate which may be produced by the action of silica. The polarizing microscope reveals excessive amounts of exceedingly fine, doubly refractile particles with relatively few of the large acicular and needle-like fragments which are so common in the ordinary silicotic lesions. In the case of the 2 white tunnel workers who admitted having had 4 to 7 years previous employment in coal mines, the silicotic nodules are 4 or 5 times as large and frequently occur as aggregations surrounded by a capsule of cellular connective tissue.

In all cases the interalveolar septa are generally thickened. In the presence of chronic infection it is impossible to estimate how much of this change is due to the silica, but when the infection is acute and exudative it probably has little effect upon the pulmonary framework. The bronchial lymph nodes were available for examination in 13 of the cases, and among these only 61 per cent showed definite silicotic nodules. In each instance at least 3 different nodes were examined.

In all of the cases death was due to pulmonary infection; in 11 this was definitely tuberculous; in 2 there was an acute and chronic non-tuberculous pneumonia, and in the other 3, tuber-

culosis was probably the cause although this could not be definitely proved.

The most common lesion was a diffuse caseous pneumonia without definite tubercle formation. The air spaces were filled with necrotic exudate which in occasional areas could still be identified as consisting of a mixture of mononuclear and polynuclear leucocytes. In many instances the pulmonary framework and even the silicotic nodules were involved in the necrotizing process. Frequently great numbers of cholesterol crystal clefts were observed. (The cleft was formerly occupied by a crystal which was dissolved in the preparation of the section.) In 5 instances the tuberculosis occurred in small broncho-pneumonic patches with caseation scattered here and there throughout the lung. Cavity formation was observed in 2 of these cases but in the other 3 only sections of the lung were obtained without record of conditions in the remaining portions. Tubercle bacilli were rare in all instances, and in some of the obviously tuberculous lesions none could be found.

There were 2 cases (Nos. 63, 71) in which the infectious process was presumably tuberculous but a definite diagnosis could not be made. The air spaces were filled with a partially necrotic exudate and some of the silicotic nodules had a central zone of granular necrosis but neither bacilli nor typical tuberculous lesions could be discovered.

The acute and chronic non-tuberculous pneumonias were interesting. One case (No. 64) occurred in a white man with 3 or 4 years' experience in a coal mine who showed extensive silicosis of the small nodule type and considerable anthracosis. In one lung the air spaces between the silicotic nodules were overdistended with an exudate consisting almost entirely of polynuclear leucocytes with numerous pneumococcus-like organisms; in the other there was organization with extensive interstitial fibrosis and an acute exudate in the distorted air spaces. The other case (No. 34), a more acute unresolved pneumonia also occurred in a white man, a sand blaster for 17 months. His symptoms appeared at the end of this period but he worked at another job in the same plant for 17 months more before he was forced to enter a hospital. He died 4 months later. The roentgenological and pathological diagnosis at this institution was silicosis complicated by tuberculous broncho-pneumonia and right heart failure. At first sight the sections did suggest an element of tuberculosis but further study has convinced the writer that the infection was not of this character. Silicotic nodules were embedded in areas of massive fibrosis from which most of the air spaces had disappeared or were represented by distorted slits. Other less involved portions of the lung showed air spaces with thick walls and a mixture of acute and organizing fibrinous exudate in their lumina. In still other areas edema and passive congestion dominated the picture.

The gross appearances have not been described because they were uninformative. In only 8 of the tunnel workers was the author able to see the whole lungs and in them the tissue had already been fixed in formalin; from the remaining ones large blocks of tissue were available for study. In 7 of the group caseous tuberculous pneumonia was so extensive that it obscured any other lesion which might have been present. Careful search with a lens disclosed nodules not greater than 1 mm. in diameter which suggested a complicating early silicotic reaction in 2 instances. In 2 others the diagnosis was questionable. Had the occupational histories not been known, it is doubtful whether silicosis would have been thought of in any of them. Careful inspection of the apices of the lungs failed to disclose evidence of healed tuberculous lesions, and no calcified foci could be detected in the lymph nodes.

Dr. Clayton S. Smith [5] has analyzed portions of the lungs of the tunnel workers for silica. The complete details of his study are published elsewhere in this *Journal,* but he has permitted the author to summarize his findings in this report. He made separate analyses of various portions of each lung but in Table I his figures for each case have been averaged. The percentage of silica in the dried lung varied between 0.84 and 3.1. It was lowest in the case with no silicotic nodules (No. 69). A second case with very slight lesions (No. 66), showed only 0.875 per cent. In the others, with variable amounts of reaction, it ranged between 1.24 and 3.1, but the amount was not proportional to the period of

employment. The highest figure occurred in a man with 3 or 4 years' previous employment in a coal mine, but the other coal miner who worked 7 years at the same occupation showed less silica than most of the men exposed only in the tunnel. For the sand pulverizers Dr. Louis Gershenfeld furnished reports of analyses which showed from 1.8 to 2.5 per cent of silica in the dry lung tissue. The duration of employment reported for this group seems most untrustworthy.

DISCUSSION

It is indisputable that this group of workmen exposed in three different occupations to high concentrations of silica dust had developed the histological lesions of silicosis by the time of their death. Whether their exposures were actually as short as 8 to 17 months cannot be established with certainty, but their age would preclude any occupation of many years duration. Dr. Smith's analysis of the lung tissues of the tunnel men showed 1.24 to 3.1 per cent of silica in the dry weight of the lungs with histological lesions of silicosis. In the one man with no specific changes his figure was 0.84 per cent. For the sand blasters the corresponding figures varied from 1.81 to 2.45 per cent. These are as high as McCrae's [6] percentages for ordinary silicosis in South African gold mines. He does not state the duration of exposures in his report but the per cent of silica in 6 dried lungs varied from 1.39 to 4.47 with an average of 2.63. The precise amount of reaction which had developed at the end of the period of employment is unknown as no case came to autopsy at that time. All observations were made from 9 to 20 months later when the men had died of infection. Within this period some change in the lesions would be expected particularly in the presence of infection. The significant feature is that the gross anatomical changes which had developed at the time of death were not sufficiently extensive or characteristic to be recognized as silicotic in origin.

Strictly speaking, the first question proposed in this investigation has been answered in the affirmative; silicosis can develop under these conditions of exposure but the reaction is of microscopic proportions. Since the men could not have been employed for very long periods because of their ages, and since chemical analysis showed that their lungs contained excessive amounts of silica, it must be assumed that this material accumulated within a comparatively short time.

The second question was whether the silicosis developing under such conditions in any way differed from the usual picture observed in miners, quarrymen, founders, etc. Direct comparison is impossible for lack of pathological material on men dying after short periods of employment in these industries. It is quite possible that the early lesions of the more familiar type of the disease are comparable both in rate of development and character to those described for this series of cases which had been called "acute." In the absence of any other basis of comparison for the rate of development of silicotic nodules, reference is made to animal experiments.[7] In guinea pigs exposed for from 1 to 2 years to concentrations of approximately 200 million pure crystalline silica particles per cubic foot of air, the number of nodules per unit area was much smaller than that in the human cases under discussion. After 1 year's exposure both were approximately of the same size but the human lesions were completely hyalinized, whereas in the animals this change was only beginning to appear. During the ensuing year the total area of the experimental nodule attained approximately the same size as the central hyaline zone in the nodule of the sand blasters and tunnel

men exposed for similar periods. Whether the difference is a reflection of variation in the reaction to silica in the two species or whether it signifies that the exposures in the human cases were greatly in excess of the 200 million particles per cubic foot of air cannot be ascertained.

The character of the silicotic lesions encountered in these cases differs in three particulars from those usually observed:

1. The individual nodules are small but they are massed in zones of considerable width along the course of the deep and superficial pulmonary lymphatic trunks and they are embedded in wide bands of connective tissue which in many places has itself become hyalinized. In the ordinary case the nodules are much larger, sometimes developing from a single center and sometimes resulting from the fusion of adjacent foci; but they tend to remain discrete and in the absence of concurrent infection they are not matted together in a mass of fibrous tissue.

2. The lymph nodes which drain the lung are ordinarily severely affected before the pulmonary changes have progressed very far, but in about one-third of the cases under discussion there was no evidence of silicosis in this location; and in others the involvement was relatively slight. It is possible that this may have been due to faulty selection of material, but it seems rather unlikely, for in most instances several nodes were sectioned.

3. All of the lungs showed an appreciable generalized thickening of the alveolar walls. Some of this reaction was undoubtedly due to the superimposed infection, but in the acute caseous pneumonias such proliferative changes are absent or not at all prominent.

The objection will immediately be raised that the unusual features of the silicosis which have been described are only unusual because of the coexistent infection. However, it should be remembered that the caseous pneumonias, at least, were very acute processes, probably non-existent during the period when these men were at work and the primary reactions to the dust were taking place. The group was composed of young adults, negroes in most cases, with probably little or no immunity to tubercle bacillus, so that their infections ran an acute course with a fatal termination in 15 months or less. If they had become infected while their silicosis was in its formative stages they would not have been able to continue so strenuous an employment and many would have died months earlier. It is tempting to speculate that because their microscopic silicotic lesions impaired the efficiency of their pulmonary lymphatics, these non-immune individuals were rendered even more susceptible to the action of the tubercle bacillus. In the non-immune individual with recognizable *advanced* silicosis a primary infection with the tubercle bacillus will sometimes cause death in 3 or 4 months. Whether this is also true of the type of silicosis under discussion remains to be demonstrated.

For these reasons it is believed that the localization of the silicotic nodules in this group of cases was not materially influenced by the terminal tuberculosis. Possibly their rate of development was accelerated by the presence of the infection. If it is granted that the dust concentrations to which the men were exposed were unusually high, this might well be the cause for the peculiarities described. It is known that if dust is inhaled for a long time in concentrations only moderately in excess of the physiological limits, the first manifestations of its presence will be reaction in the lymphatic system, both in the mediastinal lymph nodes and in the lungs. Such is the sequence in ordi-

nary silicosis. On the other hand, if the concentrations are so excessive that lymphatic system is entirely inadequate to eliminate the major portion of the foreign material, reaction would be expected to take place within all portions of the lung. The unusual picture of widespread thickening of the alveolar walls and of broad bands of cellular or hyaline connective tissue studded with nodules along the superficial lymphatics of the pleura and the deep lymphatics in the septa, bronchi, and blood vessels has been interpreted as reaction to excessive quantities of dust.

To what extent the factor of fineness of the dust particles contributed there is little adequate information. The sections, when examined in polarized light, exhibit exceedingly large numbers of very minute particles with comparatively few large ones. Time has been lacking to make a size distribution study of the particles from digested portions of these lungs. Experiments [8] have demonstrated that in rabbits the rate of the silicotic reaction varies directly with the size of the particles.

Finally, it should be mentioned that the diagnosis of tuberculosis superimposed upon silicosis of this character has often been most difficult. Other pathologists reviewing the same material have in some instances failed to agree. The lesions characteristic of the chronic forms of tuberculosis are often few in number. The necrosis produced by silica in non-tuberculous exudates often simulates caseation which has necessitated the examination of many sections before definite tuberculous lesions could be detected. Tubercle bacilli were only discovered after prolonged search in 50 per cent of the lesions examined. It is unfortunate that guinea pig inoculations could not have been made. If errors have been made it is felt that they occurred in classifying the non-tuberculous pneumonias as such rather than the reverse.

The roentgenographic appearances of the tunnel men have been discussed by H. L. Sampson.[9] He and the author had opportunity to examine a large number of films from other members of this group, but those of the particular men who died and were autopsied have not yet become available. In the group as a whole roentgenographic evidence of silicosis was very slight or absent; only changes due to infection could be readily recognized.

In view of the fact that microscopic examination revealed so many silicotic lesions, it is perhaps surprising that the X-ray failed to detect them. Analogous situation has been encountered in the roentgenological study of experimental silicosis in rabbits. After 13 months' exposure to rather heavy concentrations of crystalline silica, the X-ray reveals no definite change in the lung of the living animal, and yet if it is killed the lungs show a most extensive nodular silicosis. After 2 years' exposure, however, the amount of disease increases sufficiently to permit the visualization of nodular shadows. The same conditions obtain with respect to miliary tuberculosis in human beings. The lungs may show no evidence of a disseminated process in one examination, but if serial films are taken the time soon comes when tubercles can be visualized throughout the lungs.

SUMMARY

A histological study of 15 cases of so-called " acute " silicosis has been reported. This was based upon examination of the lung tissues of 9 tunnel workers exposed for 9 to 13 months, 3 sand blasters for 12 to 17 months, and 3 sand pulverizers on whom satisfactory histories of exposure were not obtained. Chemical analysis demonstrated that their lungs contained as much silica as those of South African gold miners employed for long periods. Their alleged occupational exposures did

PLATE I

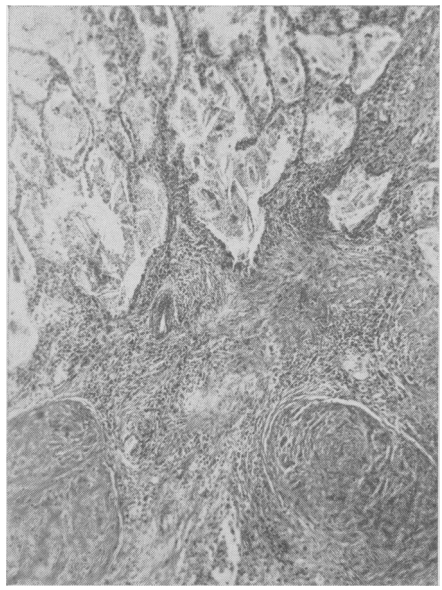


FIGURE 1.—14 months exposure in tunnel; died 13 months later. Silicotic nodules surrounded by cellular connective tissue. Exudate in air spaces.

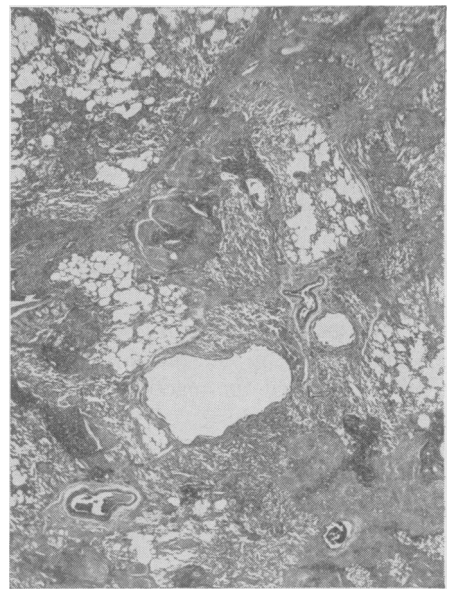


FIGURE 2.—7 years in coal dust, 13 months in tunnel; died 9 months later. Silicotic nodules larger than those in Figure 3; marked interstitial fibrosis; pneumonic reaction in lower fourth of section, probably tuberculous.

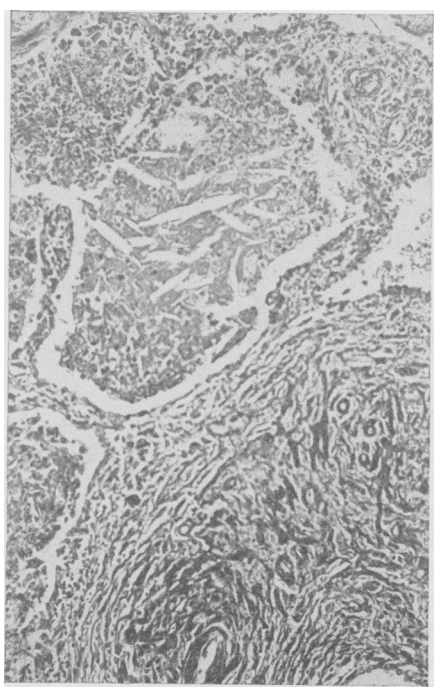


FIGURE 3.—12 months in tunnel; died within a year of acute tuberculous pneumonia. Section through area without obvious tuberculosis. Note clusters of very fine nodules along vessels through the center of the field.

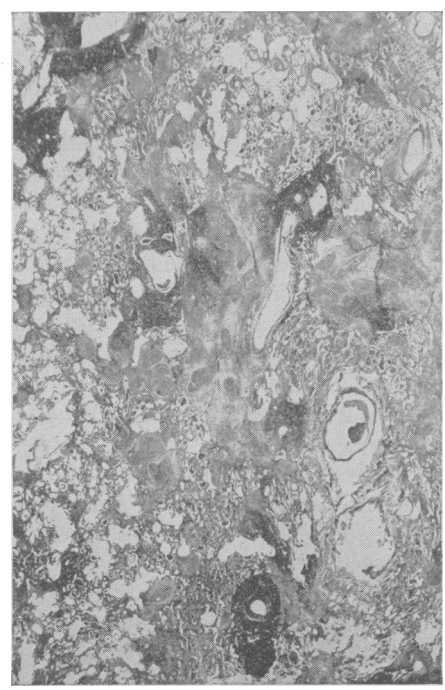


FIGURE 4.—11 months sand blasting; died 22 months later of acute tuberculous pneumonia. Silicotic nodule in right lower corner. Thickened alveolar wall and perivascular cellular fibrosis in right upper field. Necrotic exudate and cholesterol clefts within air spaces.

not exceed 17 months, a contention which is supported by the early age of the group as a whole. These men died of infection 9 to 21 months after ceasing work. In 11 of them the infection was considered to be definitely tuberculous, and in 2 others it was probably so. The other 2 had unresolved pneumonias. All but 1 of them, a tunnel man for 13 months, presented microscopic lesions characteristic of silicosis, but in no case was the disease sufficiently far advanced to be diagnosed on gross examination of the fixed tissues. The character and distribution of the changes did not resemble that usually seen in silicosis among miners and others who have died after prolonged exposures. This silicosis was characterized: (1) by masses of small nodules embedded in broad sheets of fibrous tissue surrounding the pulmonary lymphatics instead of isolated conglomerate nodules along the trunks and in the pulmonary parenchyma; (2) by a generalized fibrous thickening of the alveolar walls which is generally considered to be a late manifestation; and (3) by the absence or only slight involvement of the mediastinal lymph nodes which are usually replaced by fibrosis before extensive changes appear in the lung. These differences were interpreted as due to the inhalation of such excessive amounts of fine dust that little of it could be eliminated by the lymphatics. In most cases the tuberculous complication was so acute that it probably developed after the reaction to the dust was well established, and thus failed to influence the picture greatly.

The acuteness of the infection was explained by the fact that the majority of the men were young negroes with presumably little immunity to tuberculosis. Although there is histological evidence of silicosis, atypical in character, it seems doubtful whether there is justification for describing the process as "acute." At least this should only be done after serial roentgenograms together with post-mortem examinations have demonstrated the outcome of the allegedly heavy exposures to silica. The difficulty in properly evaluating the element of infection in the tissues suggests the need for caution in the interpretation of roentgenograms.

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